Information

Endocarditis– Clinical Findings and Management

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THE BASIC LESION of endocarditis is a friable, verrucous three-layered vegetation. The inner and thickest layer consist of fibrin, collagen and necrotic blood elements, the middle layer is made up of bacteria, and the outer layer of bacteria and loose fibrin strands. Most cases of endocarditis are due to bacteria, but fungi and rickettsiae may be responsible.

Sterile endocarditis may persist after successful treatment of infected heart valves and result in emboli. In addition, malignant disease, pulmonary emboli, pneumonia and systemic lupus erythematosus can be associated with sterile endocarditis. Emboli are at least as frequent in sterile as in bacterial endocarditis.

Before antibiotics were available, when mortality was virtually 100 percent, a clinical separation between acute and subacute endocarditis was made on the basis of duration of survival. This has little merit today, although, on occasion, fulminating endocarditis is still seen with Staphylococcus aureus or pneumococcal infection. For practical purposes, however, all cases of endocarditis should be considered as potentially life-threatening.

Rheumatic heart disease is still the most common underlying predisposing condition. Since the mitral valve is the most commonly affected in rheumatic fever, it is most commonly involved in bacterial endocarditis; next, in decreasing order, are aortic, tricuspid and pulmonic valves.

Among congenital abnormalties, ventricular septal defects are the most commonly infected, then tetralogy of Fallot, patent ductus arteriosus, pulmonary, and aortic stenosis. Endocarditis may complicate coarctation and aneurysm of the aorta, arteriovenous fistulae, syphilitic heart disease, and bicuspid aortic valves. Thrombi formed by a jet of blood streaming from a high to a low pressure area can become infected. Such a jet does not occur in inter-atrial septal defects, nor does endocarditis.

Endocarditis may occur on normal heart valves in elderly persons, and in persons infected with S. aureus, pneumococci, meningococci, or brucellae.

Bacteremia, which is generally a necessary condition to establish endocarditis, may result from dental extractions or cleaning of teeth, respiratory infections, pyoderma, prostatic massage, cystoscopy, urinary and cardiac catheterization, normal obstetic delivery, prolonged intravenous usage, or self-administration of drugs by the intravenous route—a current social problem. As a complication of surgical operation on the heart, infection introduced at the time of operation may lead to endocarditis.

When antibiotics were first introduced, the bacterial causes of endocarditis were: streptococci 62 percent, pneumococci 15 percent, staphylococci 13 percent, gonococci 7 percent, miscellaneous and multiple organisms 3 percent. Over the past two decades there has been a pronounced reduction in the occurence of endocarditis due to gonococci and pneumococci and an increase in cases due to staphylococci and group D streptococci, and in cases in which blood cultures are negative. The latter may represent either sterile endocarditis or infection with organisms such as bacteroides, listeria or microaerophilic streptococci which require specialized cultivation.

The changes in organisms are attributed to antibiotic usage, long-term stay in hospital, aging patient populations and aggressive surgical manipulation.

Clinical Manifestations

Textbook descriptions of clinical symptoms include fever, heart murmur, splenomegaly, anemia and some manifestations of peripheral embolization. In the majority of patients its manifestations are not so clear-cut. In a recent series 75 percent of patients with endocarditis had no evidence of peripheral embolization, 60 percent

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had no splenomegaly and 4 percent had no heart murmur.

Murmurs of endocarditis are generally insufficiency murmurs. Flow murmurs in patients with fever and anemia are often considered as evidence for endocarditis, but frequently these findings merely represent a hyperdynamic circulation. Rapidly changing murmurs are rarely evidence of endocarditis unless there has been rupture of chordae tendinae or papillary muscles, or heart failure. Changes in cardiac function alone characteristically produce changing cardiac murmurs. An infrequent cause of change in cardiac function is endocarditis. However, sudden onset of myocardial infarction or congestive heart failure in a patient with previously compensated congenital or rheumatic heart disease may be the presenting finding.

Fever, the most common physical finding, may be absent for a variety of reasons including the taking of aspirin, shock, advanced age, antibiotic treatment, uremia, steroid treatment, or severe infection.

Endocarditis may present as an acute fulminating illness in which meningitis, intracerebral hemorrhage, congestive heart failure, cardiac arrhythmia or uremia is prominent, and hence the underlying cause may be overlooked. Neurologic manifestations are particularly common in the elderly and in patients with cyanotic congenital heart disease. In such persons signs of brain abscess, toxic psychosis or meningitis may be the initial event. On occasion, peripheral embolization may be a presenting form, with such manifestations as infarction in fingers, toes, nose or ear lobes. Embolization of larger blood vessels suggests fungal or S. aureus endocarditis; a common site of infarction is the spleen. Splenomegaly is often absent at the onset, although patients may complain of pain in the left upper quadrant of the abdomen. Both renal embolization and glomerulitis—the latter probably autoimmune in origin—are common in cases untreated for some time. Hematuria, proteinuria and azotemia resulting from involvement of this type may lead to an initial diagnosis of glomerulonephritis or renal failure. The anemia is non-specific and is present in approximately two-thirds of the cases. It is usually normochromic and normocytic and may be associated with monocytosis.

Treatment

Antibiotics either singly or in combination should be chosen for bactericidal activity against the infecting organisms. Ideally, the antibiotic should have a sufficiently low order of toxicity to permit an appropriate duration of treatment, three weeks at least. Some authors have presented good results in the treatment of S. viridans endocarditis with large doses of penicillin and streptomycin in two weeks. Tube dilutions to determine bactericidal endpoint of antibiotics on the organism present in the patient, and the bactericidal effects of his serum upon the organism, are helpful in guiding therapy. A bactericidal blood level five times greater than the minimal *in vitro* bactericidal level is generally sought.

When blood cultures are negative or when treatment was started before appropriate cultures have been obtained, treatment may be carried out with large doses of penicillin (12 to 20 million units a day) and streptomycin (250 mg every six hours) for at least four weeks.

Antibiotic therapy will not improve the damaged valve, and distortion of valve function may follow healing and scarring. Surgical correction may be needed ultimately. In cases of severe valvular disease and cardiac decompensation, valvuloplasty may be required. Cardiac operation in the face of infection may be necessary. This should not be undertaken lightly, but may be the only recourse if infection is uncontrolled or heart failure due to valve damage is not controllable by drug therapy.